

manual pressure to tighten a loop of rubber tubing around the base of the scrotum. The rubber loop is clamped with a metal ring to hold it tight, resulting in necrosis of the entire scrotum and testicles due to ischemia. This was the first year the device had been used by this producer. Within three weeks, he noticed one of the smaller calves walking stiffly and rolling its eyes back. Within two days, an additional ten animals were showing similar signs.

Clinical examination revealed calves with intermittent prolapse of the nictitating membrane. Four developed a stilted gait and held out their tails stiffly behind them. One calf had marked salivation and could not open his mouth. All of the affected calves had been castrated, except one that had only been dehorned at processing. Tetanus was suspected. The most severely affected calf was shot and forwarded to the Airdrie Regional Animal Health Laboratory for postmortem examination, to eliminate other potential diagnoses, including rabies.

The calf had reduced ruminal contents and internal lesions associated with a gunshot wound to the chest. The scrotum was desiccated, black, and exceptionally putrid. There were no central nervous system lesions, and rabies evaluation was negative. Microscopic examination of tissues was unremarkable. Anaerobic culture of the scrotal tissue yielded heavy growth of a pure culture of *Clostridium tetani*. Smears revealed large numbers of typical "tennis racket" shaped organisms.

All calves were treated with a high dose of procaine penicillin for two days. On the second day, all calves received tetanus toxoid. Those that had undergone castration or dehorning were also given 1500 units of tetanus antitoxin. After three weeks, the three most severely affected calves had died. Two calves that had shown stiffness and prolapse of the nictitating membrane after initiation of treatment were alive and eating and drinking, but they were not expected to recover. Five calves that had shown only prolapse of the nictitating membrane after onset of treatment had recovered. No further cases occurred.

Clostridium tetani is an anaerobic spore-forming bacillus present in the soil and in animal feces. When

it contaminates deep wounds in which anaerobic conditions exist, it proliferates and produces toxins. These include a hemolysin that causes tissue necrosis, which enhances anaerobic conditions and favors bacterial growth, and a neurotoxin (tetanospasmin) (1). The tetanospasmin produces the clinical signs of tetanus. Clinical disease occurs sporadically, typically following some type of trauma that allows penetration of the organism.

Tetanus rarely occurs in western Canada. The clinical signs in this instance were dramatic and classical, especially the prolapse of the nictitating membrane. Entry of the agent probably occurred at the site of local necrosis due to the elastic, and the anaerobic environment created in the scrotum provided an ideal environment for proliferation. The metal clip that held the elastic may have contributed to local trauma through abrasion. Local seepage of toxin could have occurred despite disruption of circulation by the elastic castrator.

It is difficult to evaluate the effect of treatment on the course and outcome in this outbreak, since we cannot predict the number of animals that would have been affected without intervention. The treatment measures instituted were a considerable expense. While they may have prevented the loss of additional calves and provided the owner with the consolation that "everything that could be done was being done", this is another instance where prevention would have been more cost effective. Producers should be warned that, if they intend to use a bloodless castrator, calves must be vaccinated against tetanus and not all multiclostridial vaccines contain *Clostridium tetani*.

Reference

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British Columbia

Infectious hematopoietic necrosis in Atlantic salmon in British Columbia

In the summer of 1992, an outbreak of infectious hematopoietic necrosis (IPN) developed in farmed Atlantic salmon in British Columbia. This is a rhabdoviral disease affecting only salmon. The name derives from histopathological changes observed in diseased sockeye fry (1). Infectious hematopoietic necrosis is known to occur widely in British Columbia, particularly in sockeye salmon (2). Many populations of sockeye and kokanee salmon (*Oncorhynchus nerka*) are routinely found to have the infection at spawning. The disease can subsequently cause high mortality in fry but rarely affects older fish (2).

Increasing mortality was observed in a group of farmed Atlantic salmon smolts (about 1 year old),

approximately six weeks following transport from fresh to salt water. Affected fish were dark, lethargic, and swam slowly near the surface. Gills were pale brown. Fish opened for postmortem had pinpoint hemorrhages on peritoneal surfaces, in pyloric cecal fat, and in the lateral line and dorsal sinus area of skeletal musculature. Patchy hemorrhage in the kidney was a frequent finding. Histopathological changes consisted of endothelial cell necrosis in multiple tissues, leading to petechial and ecchymotic hemorrhages throughout the body. Fresh kidney samples inoculated onto chinook salmon embryo cell line-214 (CHSE-24) cells produced cytopathic effects. The isolated virus was determined to be a rhabdovirus by glutaraldehyde

fixation followed by phosphotungstic acid staining. Subsequent serum neutralization and neutralization index tests with antiserum supplied by the Pacific Biologic Station, identified the virus as that causing IHN. An intercurrent systemic infection with *Aeromonas hydrophila* was detected by bacterial culture of spleen and kidney.

Mortalities in the first pen affected peaked after about 17 days and then dropped back to normal by seven to eight weeks after the start of the outbreak. Mortality was lower in pens medicated with oxytetracycline, possibly because secondary intercurrent *A. hydrophila* infections were treated. The disease showed a similar pattern in adjacent pens, with mortality on the farm eventually returning to normal levels in all pens.

This outbreak is the first occurrence of infectious hematopoietic necrosis in Atlantic salmon in a salt water netpen in British Columbia. The circumstances of the disease strongly suggest that infection was transmitted to the fish after they entered salt water, possibly during well-boat transportation or after transfer to the netpen. The group of fish experienced

severe stress during transport and seawater introduction, and this situation would have helped the virus to overcome fish defenses. Viruses may have been transmitted to the farmed fish through sealice infections acquired from migrating maturing sockeye. Ectoparasites have been shown to be transport hosts in freshwater (3).

References

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